



## Optimal Control of Transmission Dynamics of Meningitis Disease with Vaccination, Campaign, and Treatment Factors

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### ABSTRACT

Meningitis disease is an inflammation of the membranes that protect the brain and spinal cord. This disease can be fatal to life-threatening because symptoms can appear suddenly. Meningitis is also common in infants and young children and can cause complications if not treated promptly. To prevent infection of susceptible compartment and to speed recovery of infected compartments, vaccination, campaign, and treatment are needed to prevent the spread of the disease or at least reduce the number of carrier and infected individuals. Therefore, we propose a model of meningitis disease spread consisting of five compartments, namely susceptible, carrier, infected with and without symptoms, and recovered. Vaccination, campaign and treatment are included in the model mechanism as controls. This study aims to observe the effectiveness of vaccination, campaign, and treatment in inhibiting the spread of the disease and accelerating the recovery of infected individuals. The Pontryagin minimum principle is followed to derive the optimal control problem. Numerical simulation using Runge-Kutta of order four scheme with the forward-backward sweep approach is applied to visualize the curves of state and control variables. From the numerical simulations, it is found that vaccination, campaign, and treatment are reasonable to apply in order to bridle meningitis disease from the population.

## 1. Introduction

Public health is concerned with preventing diseases from occurring and preventing diseases from becoming epidemics. Some diseases that are still of concern to the world are kidney disease, Covid-19, and breast cancer [1-3]. The application of mathematical models as an approach to understanding the spread of disease has been widely used in various aspects of the spread of diseases. Researchers studied some ways, like vaccination, fogging, treatments, and so on, to prevent the epidemics. Wiraningsih *et al.*, [4,5] have investigated the spread of rabies and used vaccines and treatment as efforts to prevent the spread of the disease. The dynamics of malaria transmission using a variant of the SIR model has also been studied in [6] by considering migration in the model. Vaccination and spraying insecticides as efforts to overcome the spread of malaria have been studied in [7]. Muin *et*

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*al.*, [8] reviewed the development of the SEIR model to analyze the spread of hepatitis by using vaccines and treatment.

Meningitis is inflammation of the meninges, which is the membrane surrounding the spinal cord and brain. Bacterial meningitis is generally caused by germs like *Listeria monocytogenes*, *Streptococcus pneumoniae*, Group B *Streptococcus*, *Neisseria meningitidis*, and *Haemophilus influenzae* [9]. Meningitis spreads from one person to another if there is close contact and occurs for a long time with a carrier or infected individual through coughing or sharing personal items contaminated by the bacteria. Bacterial meningitis is characterized by severe headaches and fever, vomiting, discomfort with bright light, and a stiff neck, which results in seizures, delirium, and death [10]. Meningitis will get worse if symptoms are not detected early, and if not given the right treatment, the individual can die [9]. Almost all bacteria that enter the meninges can cause meningitis. Bacterial meningitis is sometimes caused by infections in other parts of the body (lungs, ears, nose, throat, and sinuses) that spread to the meninges [11]. The combination of environmental effects, hosts, and organisms can cause epidemics. Besides the immunological aspect of the population, extreme climatic conditions (dry season, dust storm), and acute respiratory infections also provide a separate portion of the occurrence of the epidemics [12]. Bacterial meningitis is only found in humans and is transmitted from one person to another [13]. This infectious disease affects about 1.2 million people worldwide and causes 135,000 deaths annually. It is estimated that meningococcal meningitis causes more than 10,000 deaths each year in Sub-Saharan Africa [14]. Asamoah *et al.*, [15] considered antibiotics to model the spread of bacterial meningitis with a non-linear recovery rate, and the analyses provided a potential framework for controlling the spread of the disease.

The main characteristics of the *Neisseria meningitidis* bacterium are carriers that can accommodate these bacteria in the nose and throat without any symptoms and play an important role in the spread of meningococcal disease. Most of the infected cases are obtained through exposure. When the bacteria in the infected compartment start to flood the defences of body, infection can spread through the bloodstream to the meninges [9]. Meningococcal meningitis caused by the *Neisseria meningitidis* bacterium is interesting to study because it has great potential to cause epidemics [16]. In 1987 and 2000, meningococcal meningitis was affecting pilgrims in Saudi Arabia, and in the same year, there were 99 cases of meningitis affecting Indonesian pilgrims, and 40 of them died [17]. Prevention of bacterial meningitis can be done through vaccination and preventing contact with infected people. Vaccination is the most effective way to protect children from several types of bacterial meningitis [10]. Campaigns can increase public awareness about the dangers of meningitis. Thus, the community can implement a healthy lifestyle so that it can reduce the risk of contracting the disease. Meningitis is a medical emergency, and all doctors who provide acute medical care need a good understanding of the priority of treating patients with suspected meningitis for several hours at the beginning of patient care [18]. A deterministic model studied for the dynamics of disease transmission combines vaccination in the suspected compartment and appropriate treatments in the infected compartment as controls [10,19]. The dynamics of meningococcal meningitis in nine countries in Africa using time series analysis and wavelet methods was studied in [20]. The results of their study indicated that international cooperation in the field of public health is needed to control this infection.

Mathematical models have been deployed to inform the effectiveness of health policies. It aims to help people increase their understanding of the spread and control of infectious diseases. The spread of meningitis in [9] used discrete mathematical models. It was based on cellular automata, where the population was divided into five classes: susceptible, carriers, asymptomatic infected, infected with symptom, recovered, and died. The study in [21] also investigated the spread of disease

and divided the population into five compartments: susceptible, vaccinated, carrier, infectious, and recovered. The model was then completed using the optimal control theory to determine the optimal strategy to reduce the spread of the disease by including vaccinations and using face masks. The model parameterized the meningitis outbreak using data from 2017 in Africa.

The meningitis vaccine is given to stimulate the body's immune system to make antibodies and fight the bacteria that cause meningitis. Treatment for meningitis usually varies depending on the cause; for example, antimicrobial drugs may be given for meningitis caused by bacteria. Campaigns about meningitis usually focus on the effects and consequences that a person can experience after having meningitis, as well as the impact on family members of patients who die after treatment for meningitis. In this article, a model of the spread of meningitis disease is developed in which the population is divided into five compartments: susceptible, carrier, infected with and without symptoms, and recovered. As the efforts to control the disease, vaccination, campaign, and treatment are considered in the model, the Pontryagin minimum principle is applied to minimize the carrier and infected compartments. Numerical simulations are given to visualize the curves of state and control variables.

## 2. Model formulation and analysis

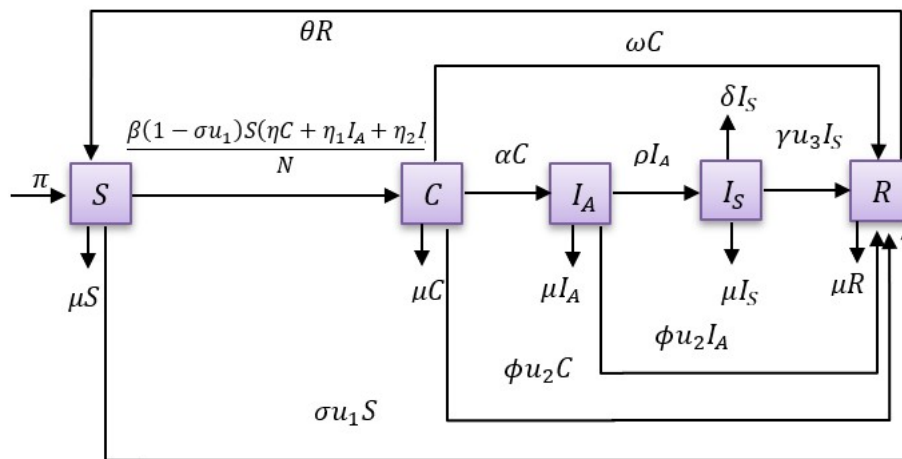
The population on the dynamics of transmission of meningitis meningococcal is commonly divided into four compartments. Some authors have considered compartments such as susceptible ( $S$ ), carrier ( $C$ ), infected ( $I$ ), and recovered ( $R$ ) in the dynamics of transmission of meningitis disease, for example, see [10,14]. The study in [12] has extended the transmission of meningitis into five compartments by differentiating the infected with symptom ( $I_S$ ) and without symptom ( $I_A$ ). This study also included optimal control problem. In this study, we assume that the newly born and migrating individual are healthy and then enter susceptible compartment with the rate  $\pi$ . The vaccinated susceptible has temporary immune system and then moves to the recovered compartment with the rate of  $\sigma u_1 S$ . The constant  $\beta$  is the rate of effectiveness contact of carriers, infected without symptoms, or infected with symptoms in the susceptible compartment. The force of new infections is given by  $\frac{\beta(1-\sigma u_1)S(\eta C + \eta_1 I_A + \eta_2 I_S)}{N}$ . Carrier compartment is healthy people who bring meningococcal bacteria in the nose and throat for a period of time, some weeks or even some months, without any symptoms and can be transmitted to others. Some studies indicated that the carrier may recover naturally from the infection without treatment and denoted such natural recovery rate as  $\omega$ .

In this study, it is also assumed that the campaign is given to the carrier compartment to increase people's awareness to carry out routine tests for meningitis. Through this campaign, some people in carrier compartment may enter the recovered compartment with the rate of  $\phi u_2 C$ . When bacteria have invaded the defences of the body, the infection can spread through the bloodstream to the meninges, so the carrier people can move to the compartment  $I_A$  with the rate of  $\alpha$ . Symptoms of the disease can appear within 2-10 days after exposure, usually within 5 days, and then move to the compartment  $I_S$  with the rate of  $\rho$ . It is also assumed that the effects of the given campaign be able to influence the people to check their health in health centre so that the risk of meningitis can be reduced before symptom of infection appear with the rate of  $\phi u_2 I_A$ . The infected compartment with symptom is assumed to have no natural recovery unless it is given accurate treatment, then it moves to recovered with the rate of  $\gamma u_3 I_S$ . It is also assumed that the infected compartment with symptoms dies cause by disease with the rate of  $\delta I_S$ . From the epidemiological perspective, the people who recover do not get permanent immunity and then again become susceptible with the

rate of  $\theta$ . Based on the given assumptions, we construct the mathematical model of transmission of meningitis disease with vaccination, campaign, and treatments as denoted by Eq. (1) and Figure 1.

$$\begin{aligned} \frac{dS}{dt} &= \pi + \theta R - \frac{\beta(1 - \sigma u_1(t))S(\eta C + \eta_1 I_A + \eta_2 I_S)}{N} - (\mu + \sigma u_1(t))S \\ \frac{dC}{dt} &= \frac{\beta(1 - \sigma u_1(t))S(\eta C + \eta_1 I_A + \eta_2 I_S)}{N} - (\mu + \omega + \alpha + \phi u_2(t))C \\ \frac{dI_A}{dt} &= \alpha C - (\mu + \rho + \phi u_2(t))I_A \\ \frac{dI_S}{dt} &= \rho I_A - (\mu + \gamma u_3(t) + \delta)I_S \\ \frac{dR}{dt} &= \sigma u_1(t)S + (\omega + \phi u_2(t))C + \phi u_2(t)I_A + \gamma u_3(t)I_S - (\mu + \theta)R \end{aligned} \tag{1}$$

The total population is denoted by  $N = S + C + I_A + I_S + R$ . Since we consider a compartment as a group of people, then the initial conditions of each compartment satisfy  $S(0) > 0$ ,  $C(0) \geq 0$ ,  $I_A(0) \geq 0$ ,  $I_S(0) \geq 0$ , and  $R \geq 0$ . The symbols  $\frac{dS}{dt}$ ,  $\frac{dC}{dt}$ ,  $\frac{dI_A}{dt}$ ,  $\frac{dI_S}{dt}$ , and  $\frac{dR}{dt}$  define the growth rate of susceptible, carrier, infected with symptom, infected without symptoms, and recovered compartments, respectively.



**Fig. 1.** Transmission scheme of meningitis with vaccination, campaign, and treatment

### 3. Optimal control problem

In the dynamics of transmission of meningitis disease, we focus on controlling the effects of vaccination, campaign, and treatment as the variables to minimize the accumulation of carrier compartment, infected with and without symptom compartments for a period of time. The control variable  $u_1(t)$  with  $0 \leq u_1(t) \leq 1$ . The effectiveness of vaccination in minimizing the meningitis disease is denoted by  $\sigma$ , the vaccine is assumed to be not perfect so that it has a failure rate

of  $(1 - \sigma u_1(t))$ , where  $0 < \sigma < 1$ . The control variable  $u_2(t)$  with  $0 \leq u_2(t) \leq 1$ , and effectiveness of campaign in minimizing the disease is denoted by  $\phi$ , where  $0 < \phi < 1$ . The control variables  $u_3(t)$  with  $0 \leq u_3(t) \leq 1$ , and effectiveness of treatments is denoted by  $\gamma$ , where  $0 < \gamma < 1$ . The three control variables are then set as

$$U = \{(u_1(t), u_2(t), u_3(t)) | 0 \leq u_1 \leq 1, 0 \leq u_2 \leq 1, 0 \leq u_3 \leq 1, t \in [t_0, t_f]\}$$

The objective of optimal control problem is to minimize the accumulation of carrier ( $C$ ), infected without symptoms ( $I_A$ ), and infected with symptoms ( $I_S$ ) and also minimize the cost of vaccination, campaign, and treatment. Mathematically, the objective function is given in Eq. (2).

$$J = \min_{(u_1, u_2, u_3)} \int_{t_0}^{t_f} \left[ A_1 C(t) + A_2 I_A(t) + A_3 I_S(t) + \frac{A_4}{2} u_1^2(t) + \frac{A_5}{2} u_2^2(t) + \frac{A_6}{2} u_3^2(t) \right] dt, \quad (2)$$

which subjects to Eq. (1). The constants  $A_i, i = 1, \dots, 6$  are weighted for each compartment and control variables. In other words, Eq. (2) represents finding out the optimal control variables  $(u_1^*, u_2^*, u_3^*) \in U$  such that  $J(u_1^*, u_2^*, u_3^*) \leq J(u_1, u_2, u_3)$  for every  $(u_1, u_2, u_3) \in U$ .

From Eq. (2), we need to find out the extremal  $u^*$  that satisfies  $J(u^*) = \min\{J(u) : u \in U\}$ . In order to get the extremals  $u^*$ , we follow the Pontryagin minimum principle [22,23]. Then, we define the Hamiltonian function  $H(t, x, u, \lambda) = f(t, x, u) + \lambda^T(t)g(t, x, u)$ , where  $f(t, x, u) = A_1 C(t) + A_2 I_A(t) + A_3 I_S(t) + \frac{A_4}{2} u_1^2(t) + \frac{A_5}{2} u_2^2(t) + \frac{A_6}{2} u_3^2(t)$ ,  $g(t, x, u)$  refers to Eq. (1), and the Lagrange multiplier  $\lambda = (\lambda_1 \lambda_2 \lambda_3 \lambda_4 \lambda_5)^T$ . Therefore, we write the Hamiltonian function as

$$\begin{aligned} H = & A_1 C + A_2 I_A + A_3 I_S + \frac{A_4}{2} u_1^2 + \frac{A_5}{2} u_2^2 + \frac{A_6}{2} u_3^2 \\ & + \lambda_1 \left( \pi + \theta R - \frac{\beta(1-\sigma u_1(t))S(\eta C + \eta_1 I_A + \eta_2 I_S)}{S+C+I_A+I_S+R} - (\mu + \sigma u_1(t))S \right) \\ & + \lambda_2 \left( \frac{\beta(1-\sigma u_1(t))S(\eta C + \eta_1 I_A + \eta_2 I_S)}{S+C+I_A+I_S+R} - (\mu + \omega + \alpha + \phi u_2(t))C \right) \\ & + \lambda_3 (\alpha C - (\mu + \rho + \phi u_2(t))I_A) + \lambda_4 (\rho I_A - (\mu + \gamma u_3(t) + \delta)I_S) \\ & + \lambda_5 (\sigma u_1(t)S + (\omega + \phi u_2(t))C + u_2(t)I_A + \gamma u_3(t)I_S - (\mu + \theta)R) \end{aligned}$$

Based on the Pontryagin minimum principle, we have conditions from the state variables as  $\dot{x} =$

$$\frac{\partial H}{\partial \lambda} = \left( \frac{\partial H}{\partial \lambda_1} \quad \frac{\partial H}{\partial \lambda_2} \quad \frac{\partial H}{\partial \lambda_3} \quad \frac{\partial H}{\partial \lambda_4} \quad \frac{\partial H}{\partial \lambda_5} \right)^T, \text{ which can be written in the form}$$

$$\dot{x} = \begin{pmatrix} \dot{S} \\ \dot{C} \\ \dot{I}_A \\ \dot{I}_S \\ \dot{R} \end{pmatrix} = \begin{pmatrix} \pi + \theta R - \frac{\beta(1-\sigma u_1(t))S(\eta C + \eta_1 I_A + \eta_2 I_S)}{S+C+I_A+I_S+R} - (\mu + \sigma u_1(t))S \\ \frac{\beta(1-\sigma u_1(t))S(\eta C + \eta_1 I_A + \eta_2 I_S)}{S+C+I_A+I_S+R} - (\mu + \omega + \alpha + \phi u_2(t))C \\ \alpha C - (\mu + \rho + \phi u_2(t))I_A \\ \rho I_A - (\mu + \gamma u_3(t) + \delta)I_S \\ \sigma u_1(t)S + (\omega + \phi u_2(t))C + \phi u_2(t)I_A + \gamma u_3(t)I_S - (\mu + \theta)R \end{pmatrix}$$

The conditions for costate variables are given by

$$\dot{\lambda} = -\frac{\partial H}{\partial x} = \left( -\frac{\partial H}{\partial S} \quad -\frac{\partial H}{\partial C} \quad -\frac{\partial H}{\partial I_A} \quad -\frac{\partial H}{\partial I_S} \quad -\frac{\partial H}{\partial R} \right)^T = (\dot{\lambda}_1 \quad \dot{\lambda}_2 \quad \dot{\lambda}_3 \quad \dot{\lambda}_4 \quad \dot{\lambda}_5)^T, \text{ where}$$

$$\begin{aligned} \dot{\lambda}_1 = & (\lambda_1 - \lambda_2) \frac{\beta(1 - \sigma u_1(t))(\eta C + \eta_1 I_A + \eta_2 I_S)}{S + C + I_A + I_S + R} + \lambda_1(\mu + \sigma u_1(t)) - \lambda_5 \sigma u_1(t) \\ & + (\lambda_2 - \lambda_1) \frac{\beta(1 - \sigma u_1(t))S(\eta C + \eta_1 I_A + \eta_2 I_S)}{(S + C + I_A + I_S + R)^2} \end{aligned}$$

$$\begin{aligned} \dot{\lambda}_2 = & -A_1 + (\lambda_1 - \lambda_2) \frac{\beta(1 - \sigma u_1(t))S\eta}{S + C + I_A + I_S + R} + \lambda_2(\mu + \omega + \alpha + \phi u_2(t)) - \lambda_3 \alpha \\ & + (\lambda_2 - \lambda_1) \frac{\beta(1 - \sigma u_1(t))S(\eta C + \eta_1 I_A + \eta_2 I_S)}{(S + C + I_A + I_S + R)^2} - \lambda_5(\omega + \phi u_2(t)) \end{aligned}$$

$$\begin{aligned} \dot{\lambda}_3 = & -A_2 + (\lambda_1 - \lambda_2) \frac{\beta(1 - \sigma u_1(t))S\eta_1}{S + C + I_A + I_S + R} + \lambda_3(\mu + \rho + \phi u_2(t)) - \lambda_4 \rho - \lambda_5 \phi u_2(t) \\ & + (\lambda_2 - \lambda_1) \frac{\beta(1 - \sigma u_1(t))S(\eta C + \eta_1 I_A + \eta_2 I_S)}{(S + C + I_A + I_S + R)^2} \end{aligned}$$

$$\begin{aligned} \dot{\lambda}_4 = & -A_3 + (\lambda_1 - \lambda_2) \frac{\beta(1 - \sigma u_1(t))S\eta_2}{S + C + I_A + I_S + R} + \lambda_4(\mu + \gamma u_3(t) + \delta) - \lambda_5 \gamma u_3(t) \\ & + (\lambda_2 - \lambda_1) \frac{\beta(1 - \sigma u_1(t))S(\eta C + \eta_1 I_A + \eta_2 I_S)}{(S + C + I_A + I_S + R)^2} \end{aligned}$$

$$\dot{\lambda}_5 = -\lambda_1 \theta + (\lambda_2 - \lambda_1) \frac{\beta(1 - \sigma u_1(t))S(\eta C + \eta_1 I_A + \eta_2 I_S)}{(S + C + I_A + I_S + R)^2} + \lambda_5(\mu + \theta).$$

The stationary conditions for control variables are given by  $\frac{\partial H}{\partial u} = \left( \frac{\partial H}{\partial u_1} \quad \frac{\partial H}{\partial u_2} \quad \frac{\partial H}{\partial u_3} \right)^T = (0 \ 0 \ 0)^T$ .

From the stationary conditions we get  $u_1 = \frac{1}{A_4} \left( (\lambda_2 - \lambda_1) \frac{\beta \sigma S(\eta C + \eta_1 I_A + \eta_2 I_S)}{N} + (\lambda_1 - \lambda_5) \sigma S \right)$ ,  $u_2 = \frac{(\lambda_2 - \lambda_5) \phi C + (\lambda_3 - \lambda_5) \phi I_A}{A_5}$ , and  $u_3 = \frac{\gamma I_S}{A_6} (\lambda_4 - \lambda_5)$ . Therefore, the optimal control variables  $u_1^*$ ,  $u_2^*$ , and  $u_3^*$  are stated by

$$u_1^* = \min \left\{ 1, \max \left\{ 0, \frac{1}{A_4} \left( (\lambda_2 - \lambda_1) \frac{\beta \sigma S(\eta C + \eta_1 I_A + \eta_2 I_S)}{N} + (\lambda_1 - \lambda_5) \sigma S \right) \right\} \right\},$$

$$u_2^* = \min \left\{ 1, \max \left\{ 0, \frac{(\lambda_2 - \lambda_5) \phi C + (\lambda_3 - \lambda_5) \phi I_A}{A_5} \right\} \right\}, \text{ and}$$

$$u_3^* = \min \left\{ 1, \max \left\{ 0, \frac{\gamma I_S}{A_6} (\lambda_4 - \lambda_5) \right\} \right\}.$$

#### 4. Numerical simulations

In this section, we are going to visualize the optimal paths of state and control variables that minimize the objective function  $J$ , Eq. (2). In order to do that, we follow the numerical method, namely the forward-backward sweep method, to plot the optimal path of state and control variables. The time interval  $[t_0, t_f]$  is divided into some sub intervals, from initial time  $t_0 = b_1, b_1, b_3, \dots, b_{N+1} = t_f$ . For the control variables, we define  $u_i = (u_{i1}, \dots, u_{in})$  where  $u_{ij} \approx u_i(b_j), i = 1,2,3$  and  $j = 1, \dots, n$ . The solutions of state variables  $x(t)$  and costate variables  $\lambda(t)$  are determined using the method of forward-backward Runge-Kutta of order four. The value of  $u_i$  is renewed at each iterations using formula  $u = (u_{old} + u_{new})/2$ , where  $u_{new}$  is found from the optimality condition  $\frac{\partial H}{\partial u} = 0$ , see [24]. For simulation, we set terminal time  $t_f = 15$  years and  $t_f = 50$  years. The parameter values used in this simulation are given in Table 1.

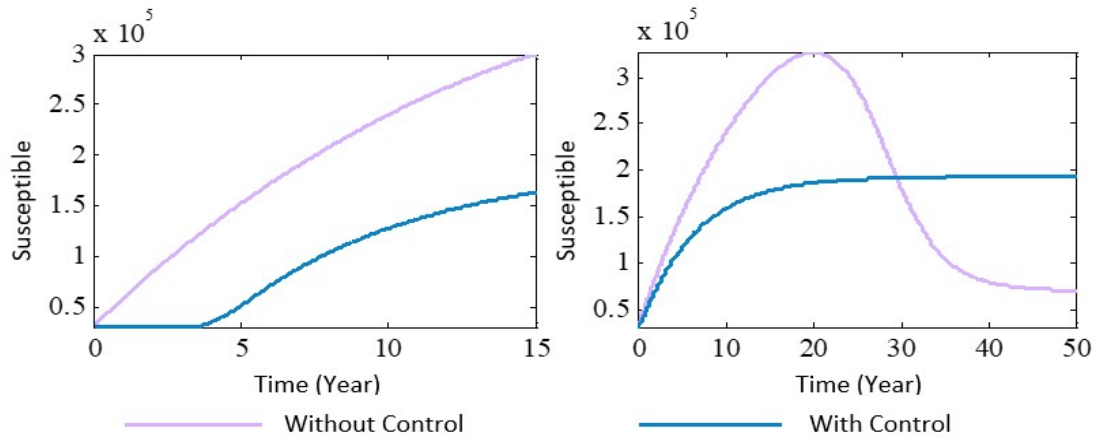
**Table 1**

Values of parameters related to the model of the spread of meningitis disease

Parameters	Description	Values	References
$\pi$	Migration and birth rate	100-100,000	[10]
$\beta$	Effective contact rate	0.88	[10]
$\eta$	Per capita infection rate by $C$	0.2-0.85	[10]
$\eta_1$	Per capita infection rate by $I_A$	0.2-0.95	[10]
$\eta_2$	Per capita infection rate by $I_S$	0.2-0.95	Assumed
$\gamma$	Effectiveness of treatment	0.1-0.9	Assumed
$\sigma$	Effectiveness of vaccination	0.85-1	[9]
$\phi$	Effectiveness of meningitis campaign	0.85-0.95	Assumed
$\alpha$	Rate of progression from $C$ to $I_A$	0.1-0.52	[10]
$\omega$	Natural recovery rate	0.06-0.2	[10]
$\rho$	Rate of progression from $I_A$ to $I_S$	0.2-0.52	Assumed
$\delta$	Disease-induced mortality	0.05-0.5	[10]
$\mu$	Natural death rate	0.02	[10]
$\theta$	Loss of immunity	0.04-2	[10]

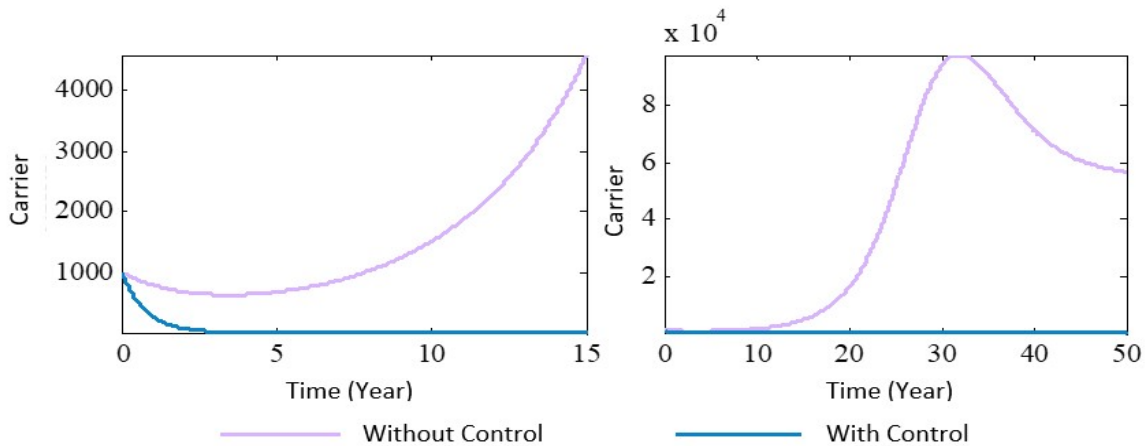
In this simulation, we set the initial values for each compartment as  $S(0) = 31,150, C(0) = 1,000, I_A(0) = 300, I_S(0) = 150$ , and  $R(0) = 468,800$ . The parameter values used are  $\pi = 10,000, \beta = 0.88, \sigma = 0.95, \phi = 0.95, \gamma = 0.95, \eta = 0.7, \eta_1 = 0.8, \eta_2 = 0.85, \alpha = 0.1, \rho = 0.2, \mu = 0.02, \omega = 0.2, \delta = 0.05$ , and  $\theta = 0.04$ . Curves of optimal variables for state and control are visualized in the Figure 2, 3, 4, 5, 6, and 7.

Figure 2 shows that in the absence of control, the susceptible ( $S$ ) initially increases and then decreases at around  $t = 20$  years, and then it tends to a certain lower value. Whereas, by giving control, the susceptible ( $S$ ) increases and then converges to a certain value. The size of the susceptible compartment will eventually be greater if given control than without being given control. This is because giving a vaccine to the susceptible ( $S$ ) will provide temporary immunity and can reduce the rate of movement to the carrier compartment ( $C$ ).



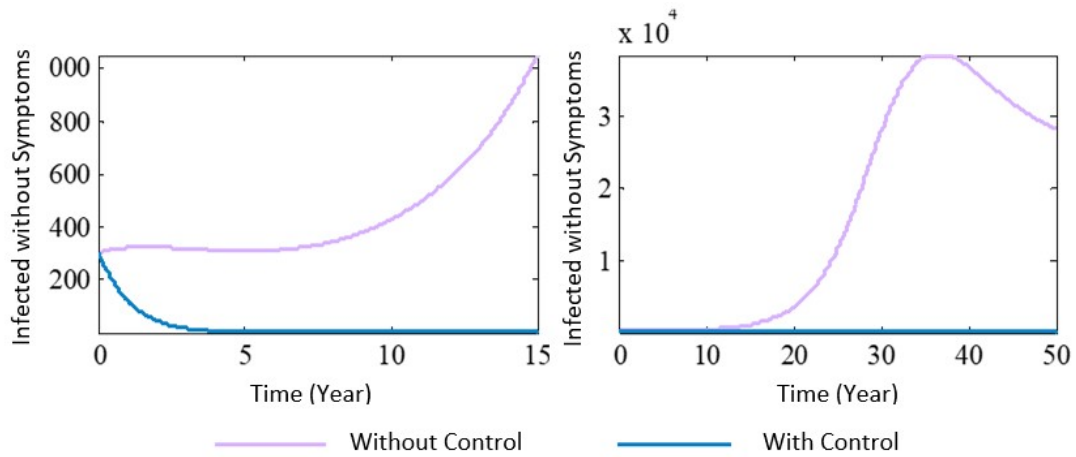
**Fig. 2.** Curves of susceptible ( $S$ ) with and without control for  $t_f = 15$  and  $t_f = 50$

Figure 3 shows that by providing controls of vaccination, campaign, and treatment on the dynamics of the spread of meningitis disease, the carrier will be depleted. This is due to increased awareness of prevention before clinical symptoms develop and people become infected. Figure 4 also shows that after controlling, the number of infected people without symptoms will decrease significantly. The effect of controls can also be seen in Figure 5, which shows that without control, the number of infected people with symptoms will increase. Meanwhile, when the control is given, the size of the infected people with symptom will decrease and then disappear. Figure 6 shows that the recovered size will be greater when given control than without control.

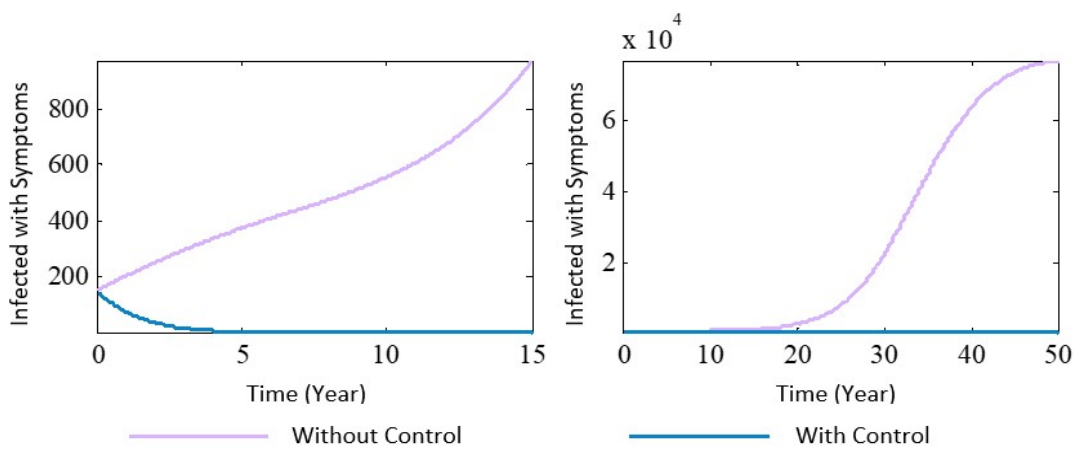


**Fig. 3.** Curves of carrier ( $C$ ) with and without control for  $t_f = 15$  and  $t_f = 50$

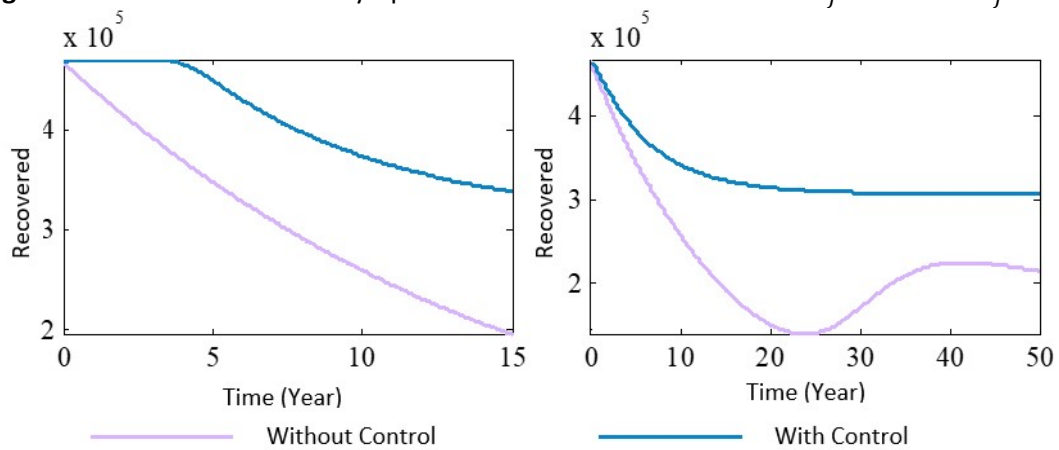




**Fig. 4.** Curves of infected without symptoms with and without control for  $t_f = 15$  and  $t_f = 50$



**Fig. 5.** Curves of infected with symptoms with and without control for  $t_f = 15$  and  $t_f = 50$



**Fig. 6.** Curves of recovered ( $R$ ) with and without control for  $t_f = 15$  and  $t_f = 50$

Figure 7 shows that the control variables  $u_1(t)$ ,  $u_2(t)$ , and  $u_3(t)$  which are initially in an effective condition, i.e., vaccination, campaign, and treatment, are given in large quantities at the beginning of time. As time goes by, the proportion of controls will continue to decrease and decrease again until the controls are no longer needed. In Figure 7, it can be seen that vaccination ( $u_1$ ) begins to decrease around  $t = 3$  to  $t = 5$ , then does not change for the next time. Campaign ( $u_2$ ) and treatment ( $u_3$ ) need to be given from the beginning, and it is seen that the proportion of control decrease around  $t = 7$ , then continues to decrease until it does not change for the next time. The compartments  $C$ ,  $I_A$ , and  $I_S$  have run out of space from the system.

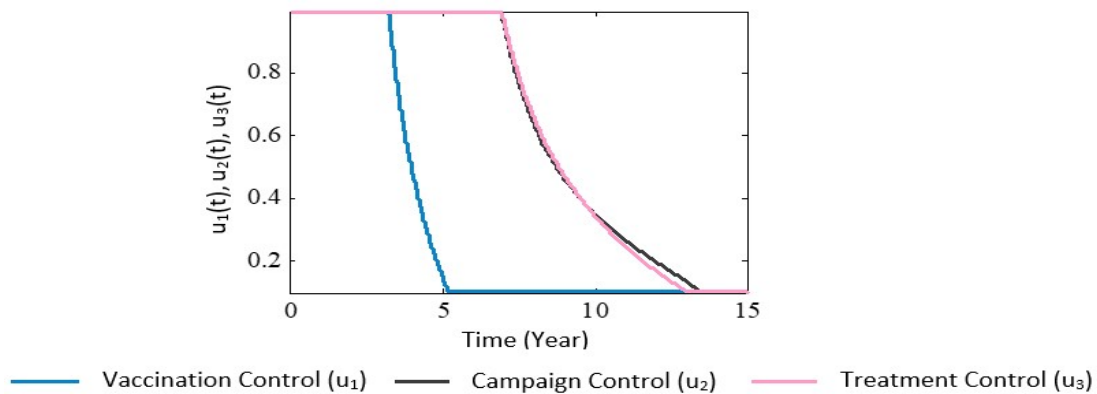


Fig. 7. Curves of control variables  $u_1(t)$ ,  $u_2(t)$ , and  $u_3(t)$  for terminal time  $t_f = 15$

## 6. Conclusions

A mathematical model of transmission of meningitis using five compartments, namely susceptible, carrier, infected with and without symptoms, and recovered by vaccine, with campaign and treatment factors, has been analyzed. Vaccination is given to the susceptible compartment to gain temporary immunity. The campaign is given to the compartments of carrier and infected without symptoms. Treatment is given to the infected compartment with symptoms. Furthermore, some efforts are made to minimize the number of carriers, infected with and without symptoms, and also to minimize costs used in the process of controlling the spread of the disease.

From the Pontryagin minimum principle, the optimal path conditions for the state and control variables are obtained. The plot of the optimal curves obtained numerically using the Runge-Kutta of order four scheme with the forward-backward sweep approach. From the simulations, it is concluded that giving vaccinations can reduce the number of susceptible to becoming carrier. Likewise, campaign for the importance of healthy living can also reduce the numbers of carrier and infected people without symptoms. Providing treatment can also reduce the number of individuals in the infected compartment with symptoms. In other words, from the simulation it is known that by introducing vaccination, campaign, and treatment accordingly, at the same time, the compartment of carrier, infected with and without symptoms, will disappear from the system.

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